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Health and Environmental Effects Profile for Cycloate

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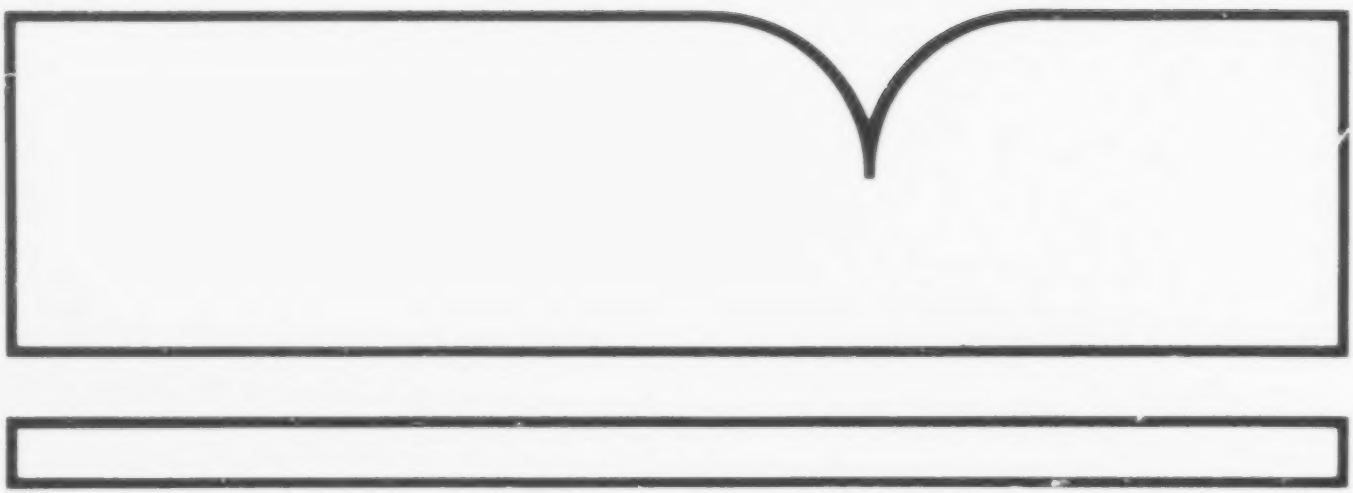

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR CYCLOATE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
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16 ABSTRACT

The Health and Environmental Effects Profile for cycloate was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for cycloate.

17 KEY WORDS AND DOCUMENT ANALYSIS

a DESCRIPTORS	b IDENTIFIERS/OPEN ENDED TERMS	c COSATI Field Group

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched."

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

ACKNOWLEDGEMENTS

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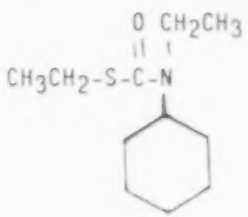
LIST OF ABBREVIATIONS

LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
ppm	Parts per million

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Cycloate is the common name for S-ethyl N-cyclohexyl-N-ethyl(thiocarbamate) (Martin and Worthington, 1977). It is also known as S-ethyl cyclohexylethylcarbamothioate (Martin and Worthington, 1977); cyclohexanecarbamic acid, N-ethylthio-, S-ethyl ester; carbamic acid, cyclohexylethylthio-, S-ethyl ester; carbamothioic acid, cyclohexylethyl-, S-ethyl ester; S-ethyl cyclohexylethylthiocarbamate; S-ethyl N-ethyl N-cyclohexyl-thiolcarbamate; eurex; and hexylthiocarbam (NIOSH, 1983); and formerly as S-ethyl N-ethylcyclohexanethiocarbamate (Martin and Worthington, 1977). This compound was introduced in the market as an herbicide by Stauffer Chemical Company under the code number "R-2063" and has a trade name of "Ro-Neet" (Martin and Worthington, 1977). The structure of cycloate is given below:



Molecular formula: $C_{11}H_{21}NOS$

Molecular weight: 215.4

The Chemical Abstract Service (CAS) Registry number for cycloate is 1134-23-2.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Cycloate is a clear liquid with an aromatic odor at ambient temperature. Some of the physical properties of cycloate, as summarized by Martin and Worthington (1977), are given below:

Boiling point:	145°C at 10 mm Hg
Vapor pressure at 25°C:	6.2×10^{-3} mm Hg

Density (temperature unspecified):	0.970 g/cm³
Solubility in water (at 22°C):	85 mg/ℓ
Solubility in organic solvents:	miscible with most solvents, including acetone, benzene, 2-propanol, kerosine, methanol and xylene

1.3. PRODUCTION DATA

Cycloate is prepared by the reaction of ethyl chlorothiolformate with N-ethyl cyclohexylamine (SRI, 1976). Cycloate is produced by Stauffer Chemical Company, Agricultural Chemicals Division, Cold Creek, AL (SRI International, 1983). Recent production data for the United States are unavailable, but the estimated yearly production of cycloate for 1971 was <1 million pounds (Lawless et al., 1972).

1.4. USE DATA

Cycloate is used as a pre-planting herbicide to control the growth of annual broad-leaved weeds and grasses and, in particular, nut grass in sugar beets and spinach (BCPC, 1977).

2. ENVIRONMENTAL FATE AND TRANSPORT

2.1. AIR

There were only limited data regarding the fate and transport of cycloate in air in the sources searched for this study (see Appendix). The important route of entry into the atmosphere is through volatilization from soil and probably from water. Gray and Welerich (1968a) reported that after surface spray application of cycloate at 3 pounds/acre on moist (15% water content) Santa Cruz loamy sand, an average value of 4.6% was volatolized in the first 30 minutes following application in three separated tests in which the greenhouse temperatures varied from 70-90°F. In a separate experiment, similar surface application of cycloate at a rate of 3.2 pound/acre resulted in vapor losses of 26% (average of two tests) and 65% at 2 and 6 hours, respectively, postapplication at a greenhouse temperature of 90°F and soil moisture content of 16-17%. In a series of experiments, these authors reported that volatilization was affected by moisture content of the soil, temperature and soil type. This is discussed further in Section 2.3.

The fate of cycloate in the atmosphere will depend upon its reactions with O_3, NO_2 and free radicals (e.g., RO•, O• and RO_2•) and its ability to undergo photolysis. Although there is no information regarding the reaction of cycloate with other atmospheric reactants, there are limited data on the photolysis of cycloate. DeMarco and Hayes (1979) exposed a hexane solution of cycloate to light from a medium pressure mercury lamp and determined an initial decomposition rate of 0.02 moles/minute from an initial concentration of 0.62 moles from an initial concentration of 0.62 moles. The products of the reaction were identified as cyclohexyl-ethyl-formamide, cyclohexylethylamine, ethyl mercaptan and diethyl disulfide. Photolysis was not affected by concentration over a range of 100-1000 ppm or

temperature over a range of 10-30°C. Although this study indicates that cycloate may undergo photolysis, the study was performed in a solution phase at a high initial concentration of cycloate. Therefore, it is possible that photolysis may not be a significant process in the ambient air under available sunlight.

2.2. WATER

There are no data on the fate and transport of cycloate in aquatic media. Based on its volatility from moist soil (Gray and Weierich, 1968a), it is likely that significant volatilization of cycloate from aquatic media will take place. Although the rates of microbial degradation of cycloate in aquatic media have not been determined, based on its biodegradability in soils (Koren et al., 1968; Gray and Weierich, 1968a; Chulakov and Zharasov, 1975), it is possible that biodegradation may play a role in determining the fate of cycloate in aquatic media.

No experimental data on the bioconcentration of cycloate by aquatic organisms are available. From binary regression equations, Kenaga (1980) predicted a bioconcentration factor of 18-50 based on the K_{oc} (soil absorption coefficient) and water solubility of cycloate (see Section 6.5.). Therefore, this compound is not expected to be significantly bioconcentrated in aquatic organisms.

2.3. SOIL

The fate and transport of cycloate in soil has been studied more extensively. The two likely processes that may determine the transport characteristics of cycloate in soil are sorption and volatilization. The major process for removal of cycloate from wet soil is volatilization. Volatilization of cycloate has been studied by Gray and Weierich (1968a), and was discussed briefly in Section 2.1. of this document. These researchers found

that volatilization of cycloate was affected by the moisture content of the soil; ~30% of the herbicide was lost in 2 hours after application to moist soil containing 17.4% moisture, whereas no detectable loss was noted within 24 hours after application to dry soil containing 1% moisture. After 24 hours, the rate of loss from moist soil decreased, probably because the soil was drying out, resulting in adsorption of the pesticide to soil particles and preventing further rapid losses. Volatilization of surface applied cycloate from dry or moist soil increased over the temperature range of 32-60°F. Temperatures >60°F decreased the rate of loss, as the soil was drying out and adsorption to soil particles occurred more readily. Gray and Weierich (1968a) also examined the effects of temperature and moisture on volatilization of cycloate from two different soil types, Felton loamy sand and Sorrento loam. Following application 3 inches below the soil surface, cycloate volatilization was 56 and 83% from Felton loamy sand and Sorrento loam, respectively, after 70 days in moist soil at 70-80°F; losses after 98 days in both types of moist soil at 40°F were much less than at the higher temperature. After 98 days in dry soil at 70-80°F, ~5% of the applied cycloate was volatilized from both soil types, as the herbicide was tightly adsorbed on the dry soils.

The volatilization and adsorption of cycloate in soils has been studied by Koren et al. (1968) and Ekler et al. (1981). Koren et al. (1968) examined adsorption and volatilization of cycloate in air-dried Staten Island peaty muck, Stockton adobe clay, Yolo clay loam, Hesperia fine sandy loam and washed sand (listed in order of decreasing organic matter content). Rapid inactivation of the herbicide occurred in the Staten Island peaty muck, due to high adsorption to organic matter (and associated microbial degredation), and in the washed sand, due to high volatilization (low

adsorption). The other three soil types slowly inactivated the herbicide, as some adsorption to soil colloids occurred that inhibited volatilization. Ekler et al. (1981) reported that cycloate was adsorbed to a high degree on meadow soil (pH 6.4; organic matter content, 3.2%) and on peat, but not on sand.

Microbial degradation of cycloate also appears to be an important mechanism for removal of cycloate from soil. In laboratory studies, cycloate was degraded more quickly in nonautoclaved soils as compared to sterile, autoclaved soils, indicating that microbial degradation was an important process in the biotransformation of cycloate (Koren et al., 1968; Gray and Weierich, 1968a). Gray and Weierich (1968a) concluded that some chemical degradation process (probably hydrolysis) also occurred, as some degradation of cycloate was also observed in sterile soil. As reported in the abstract of a Russian study, Chulakov and Zharasov (1975) observed the degradation of cycloate in South Kazakhstan soils principally by soil microorganisms that used the herbicide as an energy source.

Leaching of cycloate from soil does not appear to be an important mechanism of removal. Gray and Weierich (1968a,b) examined the possibility of cycloate leaching in Santa Cruz loamy sand, coyote sandy loam, Sorrento loam and Bower's clay; the clay content of these soils ranged from 5.0-33.2% and the organic matter content ranged from 0.8-11.6%. In all four soil types, cycloate was relatively resistant to leaching within 16 hours after application at a level of 10 pounds/acre in the top 2 inches of soil. Increasing clay and organic matter content of the soil resulted in an accompanying decrease in the amount of leaching. These results confirm the K_{oc} of 345 (experimental) and 380 (calculated), indicating only slight mobility of cycloate in soil (Kenaga, 1980).

3. EXPOSURE

STORET contains monitoring data on cycloate in water and sediments for a limited number of samples. There are 264 remarked observations for cycloate in water and 139 remarked observations for sediments collected between 1974 and 1976 in the Pacific Northwestern United States. The average concentrations of cycloate were below the detection limit, that is, <0.017 µg/ℓ for water and <14.9 µg/kg for sediments. It should be noted, however, that the STORET data do not provide specific information about the sampling site and anlytical methodology. Further, these data have not been verified, and a critical analysis of their reliability is not possible at this time. Bidnenko (1978) did not detect residues of cycloate in harvested sugar beets; however, the level of herbicide application and time to harvest were not reported in the available abstract of this Russian study.

4. COMPOUND DISPOSITION AND PHARMACOKINETICS

4.1. ABSORPTION

Aleksandrova (1975) reported rapid gastrointestinal absorption of cycloate by rats based on the detection of cycloate in the blood (time after dosing not reported) following a single intragastric dose of 1000 mg/kg. Rapid absorption following oral administration also can be inferred from the study of Aleksandrova and Klisenko (1978), maximum levels of cycloate in the tissues of rats were reached within 1 hour post-ingestion (dose level not reported). These two Russian studies were only available as abstracts and, therefore, further details regarding experimental design and absorption kinetics were not provided.

4.2. DISTRIBUTION

Only scanty, qualitative information from the abstracts of two Russian studies was available regarding the distribution of cycloate in rats. Aleksandrova and Klisenko (1978) observed the maximum levels of cycloate in tissues of rats within 1 hour post-ingestion (specific tissues affected and dose level not reported). Aleksandrova (1975) administered cycloate as a single intragastric dose of 1000 mg/kg to rats. Cycloate was detected in the blood and internal organs, with highest levels observed in the lungs, kidneys and heart.

4.3. METABOLISM

Aleksandrova and Klisenko (1978) administered an unspecified oral dose of cycloate to rats to examine the metabolic fate of cycloate. Metabolites of cycloate included cyclohexylamine and dicyclohexylamine and were detected in the liver and lungs at 1 hour after administration.

Cycloate did not undergo oxidative metabolism to form the sulfoxide metabolite when incubated <u>in vitro</u> with flavin adenine dinucleotide-dependent mono-oxygenase purified from pig hepatic microsomes and NADPH under

aerobic conditions (Hajjar and Hodgson, 1980, 1982). In contrast, Casida et al. (1975) reported that cycloate was oxidized *in vitro* to the corresponding thiocarbamate sulfoxide by the mouse hepatic microsome-NADPH system. The resulting sulfoxide was then cleaved by the hepatic soluble-glutathione S-transferase system with liberation of the mercaptan. Casida et al. (1975) also examined the *in vivo* metabolism of cycloate following intraperitoneal injection of 1 or 2 mmole/kg to Swiss-Webster mice. Neither the corresponding thiocarbamate sulfoxide nor thiocarbamate sulfones were detected in the liver as *in vivo* metabolites of cycloate. Casida et al. (1975) concluded that sulfoxidation is the initial metabolic step in the cleavage of the thiocarbamate ester group and detoxification of cycloate in mice.

4.4. EXCRETION

Pertinent data regarding the excretion of cycloate were not located in the available literature.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of cycloate were not located in the available literature. This compound is not scheduled for testing by the National Toxicology Program (NTP, 1983).

5.2. MUTAGENICITY

Kurinnyi et al. (1982) exposed mice to cycloate to determine the cytogenetic effects of the herbicide. At a minimum effective dose of 200 mg/kg, an increased number of metaphases with chromosome aberrations were observed in the bone marrow cells of exposed animals. Further details regarding route of exposure, dose levels and statistical significance were not reported in the available abstract.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of cycloate were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding the reproductive effects of cycloate were not located in the available literature.

5.5. CHRONIC TOXICITY

Pertinent data regarding the chronic toxicity of cycloate were not located in the available literature.

5.6. OTHER RELEVANT INFORMATION

The oral LD_{50} for cycloate in rats was reported to be 3160 mg/kg (NIOSH, 1983). Rebrin and Aleksandrova (1971) reported LD_{50} values of 2323 mg/kg for rats and 2285 mg/kg for mice (route of exposure not reported).

Local skin irritation was observed on rabbits following dermal application (dose number and duration not reported) of cycloate at a level of 115 or 230 mg/kg (Rebrin and Aleksandrova, 1971).

Onikienko and Blakita (1975) reported that either oral or inhalation exposure of rats to cycloate increased serum levels of a hepatic isoenzyme of lactate dehydrogenase (i.e., isoenzyme 5), indicating that liver damage may have occurred. Ostroukhova et al. (1978) reported that serum alanine aminotransferase activity was an indicator of cycloate poisoning; release of this enzyme into the serum is evidence of hepatic damage. The available abstract of these Russian studies and the NIOSH (1983) Registry did not specify further details of experimental design or observed effects.

6. AQUATIC TOXICITY

6.1. ACUTE EFFECTS

The only available information concerning the acute toxicity of cycloate was provided by Sanders (1970), who determined that the 96-hour LC_{50} was 2.6 mg/ℓ for a freshwater amphipod species, Gammarus fasciatus.

6.2. CHRONIC EFFECTS

Pertinent data regarding the chronic effects of cycloate on aquatic organisms were not located in the available literature.

6.3. PLANT EFFECTS

Pertinent data regarding the effects of cycloate on aquatic plants were not located in the available literature.

6.4. RESIDUES

Pertinent data regarding residues of cycloate in aquatic organisms were not located in the available literature.

6.5. OTHER RELEVANT INFORMATION

Kenaga (1980) used regression equations based on the water solubility and octanol-water partition coefficient of cycloate to predict bioconcentration factors in aquatic animals. The predicted bioconcentration factor was 18 based on an octanol-water partition coefficient of 345, and 50 based on a water solubility of 85 mg/ℓ.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

Under the Federal Food, Drug, and Cosmetic Act, a residue tolerance of 0.05 ppm cycloate has been established for beet roots and tops (Federal Register, 1969).

Aleksandrova and Klisenko (1982) reported the Russian maximum allowable concentration for cycloate in air of 1.0 mg/m^3.

7.2. AQUATIC

Guidelines or standards for the protection of aquatic organisms from the toxic effects of cycloate were not located in the available literature.

8. RISK ASSESSMENT

No carcinogenicity assays, chronic toxicity studies or teratogenicity studies of cycloate were located in the available literature. Cycloate increased the frequency of chromosome aberrations in the bone marrow cells of mice exposed to the herbicide (Kurinnyi et al., 1982). Onikienko and Blakita (1975) reported that either oral or inhalation exposure of rats to cycloate caused an increase in serum levels of lactate dehydrogenase isoenzyme 5, an indication of liver damage. Similarly, Ostroukhova et al. (1978) reported that another indicator of liver damage, SGPT, is affected by cyloate poisoning. The available abstracts of these Russian studies, however, provided no information regarding dose levels, duration of exposures and other toxicological endpoints evaluated.

Statements concerning available literature in this document refer to the published, quotable sources and are in no way meant to imply that confidential data, which this document could not address, are not in existence. These data, which are considered Confidential Business Information (CBI), are available in summary form as a CBI Appendix that can be accessed by individuals with appropriate clearance. Conclusions from the CBI Appendix regarding risk assessment can, however, be presented in this chapter.

The CBI Appendix to this document reviews other animal data germane to risk assessment. A tentative LOAEL of 8 mg/kg bw/day for the incidence of neuropathies and/or myopathies in rats from a chronic feeding study was presented. However, these data were inadequate for quantitative risk assessment due to the lack of the clean presentation of the incidence of histologically verifiable neuropathies and/or nyopathies in the control group.

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APPENDIX:

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

Most of these searches were conducted in February, 1983; a few were conducted March-May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

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